ELSEVIER

Sleep Medicine 2 (2001) 493–500

SLEEP MEDICINE

www.elsevier.com/locate/sleep

Original article

Psychometric comparisons of the standard and abbreviated DBAS-10 versions of the dysfunctional beliefs and attitudes about sleep questionnaire

Jack D. Edinger[a,*], William K. Wohlgemuth[b]

[a]*Psychology Service (116B), VA Medical Center, 508 Fulton Street, Durham, NC 27705, USA*
[b]*Duke University Medical Center, Durham, NC, USA*

Received 11 September 2000; received in revised form 6 November 2000; accepted 16 November 2000

Abstract

Objective: To evaluate the psychometric properties of the DBAS-10, a recently proposed abbreviated version of the Dysfunctional Beliefs and Attitudes About Sleep Scale (DBAS).

Population: Two hundred and eleven (69 normal sleepers; 142 insomnia suffers) middle-aged and older adults (age 40–79 years) drawn from two separate cohorts of research volunteers.

Method: Volunteers in the first cohort (69 normal sleepers; 69 insomnia sufferers) completed the full DBAS on one occasion. Volunteers in the second cohort (73 insomnia sufferers) completed the full DBAS prior to treatment and at multiple subsequent time points to assess treatment-related changes. A series of statistical tests were conducted with one or both cohorts to investigate the comparability of the DBAS-10 and full DBAS, the internal consistency of each instrument, the factor structure of the DBAS-10, and the validity of this instrument.

Results: Statistical findings showed that the DBAS-10 correlated highly with the full DBAS, had respectable internal consistency, effectively discriminated normal sleepers from insomnia sufferers, and detected cognitive changes resulting specifically from CBT intervention. Although factor analysis empirically identified three conceptually meaningful DBAS-10 subscales, the subscale structure varied somewhat from previous factor analytic findings with this instrument.

Conclusions: The DBAS-10 generally appears to have very acceptable psychometric properties although subscales previously proposed for this instrument may vary across research populations. Nonetheless, results encourage the use of this instrument in studies concerned with the nature and treatment of sleep-disruptive cognitions.

Keywords: Dysfunctional beliefs and attitudes about sleep; Primary insomnia; Normal sleeper; Internal consistency; Factor analysis; Cognitive–behavioral therapy

1. Introduction

Persistent primary insomnia (PPI) is a relatively prevalent and often serious form of sleep difficulty which traditionally has been attributed to such sustaining factors as conditioned arousal at bedtime and sleep-disruptive habits [1–4]. However, over the past decade, investigators have become increasing aware that dysfunctional beliefs and attitudes about sleep may underlie and support sleep-related anxiety and habits

* Corresponding author. Tel.: +1-919-286-0411; fax: +1-919-416-5832.
E-mail address: edinger.jack@durham.va.gov (J.D. Edinger).

1389-9457/01/$ - see front matter
PII: S1389-9457(01)00078-8

that disrupt the sleep process. For example, the belief that there is little one can do about poor sleep may help sustain sleep-related 'performance anxiety' whereas the belief that one should try to 'catch up' for lost sleep may lead to self-defeating compensatory practices such as remaining in bed beyond the usual rising time or subsequent daytime napping. Given this observation, Morin and colleagues [5,6] developed the Dysfunctional Beliefs and Attitudes About Sleep Scale to provide a systematic method for assessing these disruptive cognitions This scale, originally consisting of 30 items and subsequently reduced to 28 items, is composed of five rationally-derived subscales presumed to measure: (1) dysfunctional beliefs about the *consequences* of insomnia; (2) beliefs that sleep is *unpredictable and uncontrollable*; (3) unrealistic *sleep expectations*; (4) misconceptions about the *causes* of insomnia; and (5) erroneous beliefs about *sleep-promoting habits*. Preliminary research has shown that the full DBAS has highly acceptable internal consistency and several of the rationally-derived subscales reliably discriminate good/normal sleepers from poor sleepers/insomnia sufferers [5,7,8].

Despite these findings, Morin et al. [5] admit that several of their proposed DBAS subscales have fair to poor internal consistency. In addition, factor analytic studies [8,9] conducted to confirm the proposed DBAS subscale structure provided only partial support for the item clustering suggested by Morin and colleagues. Moreover, the sensitivity of the total DBAS or its component subscales for detecting pre-to-post treatment changes has yet to be documented. However, recently Espie et al. [8] identified ten DBAS items which proved highly sensitive to insomnia patients' improvements through a course of cognitive–behavioral intervention. When considered collectively, the resultant 10-item scale (DBAS-10) was found to have acceptable internal consistency (Cronbach's $\alpha = 0.69$) and yet maintain the "overall thrust of the [full] DBAS" as demonstrated by a high correlation ($r = 0.83$) between the shortened and full version of this scale. Finally, a factor analysis yielded three relatively 'pure' factors or subscales which respectively were purported to measure "beliefs about the immediate negative consequences of insomnia", "beliefs about the long-term negative consequences of insomnia", and "beliefs about the need for control over insomnia".

Give these findings, the DBAS-10 merits further study since it seems to have very acceptable psychometric properties, should save on administration time, and appears sensitive to cognitive changes occurring during insomnia treatment. The current study was conducted as a replication and extension of the work of Espie et al. [8] Specifically, we conducted this study to cross-validate the basic psychometric properties and factor structure of the DBAS-10. In addition, we tested the degree to which the DBAS-10 both discriminates insomnia sufferers from normal sleepers and reflects cognitive changes resulting from cognitive–behavioral intervention. Data for the current investigation were derived from two large funded studies concerned with the nature and treatment of PPI.

2. Methods

2.1. Study participants

Two cohorts of middle-aged and older adults recruited as research volunteers provided data for this investigation. All of these individuals were thoroughly screened via validated structured psychiatric [10] and sleep [11] interview methods, medical examination, TSH screening, and polysomnography (PSG). The first cohort consisted of 69 (34 women; 35 men) non-complaining normal sleepers (mean age 56.9 years; SD 11.7 years) and 69 (33 women; 36 men) insomnia sufferers (mean age 59.0 years; SD 10.4 years) recruited to take part in a study designed to compare laboratory and home sleep patterns. The normal sleepers in this cohort reported satisfaction with their typical sleep patterns and did not meet structured interview criteria for a sleep disorder. The insomnia sufferers had complaints of sleep onset difficulty ($n = 6$), sleep maintenance problems ($n = 27$), both onset and maintenance difficulties ($n = 31$), or non-restorative sleep ($n = 5$) for at least 6 months (mean 11.4 years; SD 9.8 years) and all met interview criteria for persistent primary insomnia (PPI). Excluded from the cohort were individuals with (a) a terminal illness; (b) a medical condition (e.g. rheumatoid arthritis, thyroid disease) that compromises sleep; (c) abnormal TSH levels; (d) a history or symptoms of psychiatric illness; (e) a history of substance

abuse; (f) sedative hypnotic dependence; (g) current use of anxiolytics, antidepressants or any other psychotropic medication; or (h) objective evidence of clinically significant (≥ 15 apneas + hypopneas/h of sleep) sleep apnea during PSG.

The second cohort consisted of 73 (34 women; 39 men) insomnia sufferers (mean age 55.5 years; SD 10.6 years) who presented to take part in a study designed to test the efficacy of cognitive–behavioral insomnia therapy (CBT) against standard progressive muscle relaxation training [12] (RT) and a quasi-desensitization placebo control [13] (PC) for treating primary sleep-maintenance insomnia. Given the purpose of the study, all of these individuals met structured interview criteria for PPI, had a mean wake time after sleep onset of at least 60 min per night as evidenced by 1 week of sleep log monitoring, and reported insomnia problems for at least 6 months (mean 13.8 years; SD 12.0 years) prior to presenting for treatment. In selecting this sample, we used exclusion criteria similar to those used in selecting the above-described cohort. However, individuals with past (but not current) symptoms of psychiatric illness were not excluded from this second cohort whereas those with PSG evidence of ≥ 15 periodic limb movement-related arousals per hour of sleep were. Otherwise the exclusion criteria used in selecting the two cohorts were identical.

2.2. DBAS questionnaire

The version of the DBAS used with both cohorts contained all 28 items listed in the Morin et al. [5] report plus three additional unscored items. Each item was accompanied by a 100-mm visual analog scale which was anchored with the words 'strongly disagree' at its far left extreme and 'strongly agree' at its far right extreme. Participants were required to draw a line through the point on the 100-mm scale which indicated their level of agreement with each item. The averaged score across the 28 scored items represented the respondents's overall DBAS score. In addition, the averaged score across the ten items composing the DBAS-10 represented the respondent's score for this shortened version of the DBAS.

2.3. Procedure

Participants comprising the first study cohort completed a variety of procedures including 6 nights of polysomnography, a Multiple Sleep Latency test, computer-administered vigilance tests, sleep logs and various questionnaires for research purposes. Each of these participants completed one administration of the DBAS during this time period. Upon completion of their study commitments, all of these individuals were compensated financially for study participation.

Those participants comprising the second cohort were asked to complete a number of monitoring procedures and questionnaires at multiple time points primarily to assess treatment-related sleep improvements. Specifically, these individuals were asked to complete objective monitoring procedures (PSG, actigraphic monitoring) and self-report measures (e.g. sleep logs, questionnaires) prior to treatment, during a 6-week treatment period, after treatment, and at a 6-month follow-up. Included among the measures administered at these time points was the full DBAS. For the purpose of this study, only the pre-treatment and post-treatment DBAS data were used in the various analyses described below.

3. Results

3.1. Correlational analyses

We first computed descriptive statistics and conducted a series of correlational analyses to assess the basic psychometric properties of the DBAS-10. In doing so, we used the DBAS data gathered from the single testing of the first cohort and the pre-treatment DBAS data obtained from the second cohort. Table 1 provides descriptive data (means, standard deviations), and results of the simple correlational analyses (Pearson's r and Cronbach's α coefficients) conducted. These data show that the DBAS-10 scores, on average, were higher and more dispersed than were the standard DBAS scores. Nonetheless, the DBAS-10 and the standard DBAS were correlated highly with each other in each group of study participants suggesting that the shortened instrument serves as an adequate substitute for the longer one. However, the alpha coefficients obtained for the full DBAS were consistently higher than those obtained for the DBAS-10. This latter finding does not seem particularly surprising since scales with more items tend to have

Table 1
Means, standard deviations, interscale correlations and Cronbach's coefficient alpha values for each group of participants

Group	DBAS			DBAS-10			*r*-Value DBAS vs. DBAS-10
	Mean	SD	Alpha	Mean	SD	Alpha	
Cohort 1: normal sleepers	26.4	9.7	0.81	33.5	14.0	0.70	0.84[a]
Cohort 1: insomnia sufferers	35.5	10.5	0.79	43.4	15.0	0.68	0.91[a]
Cohort 2: insomnia sufferers	37.7	8.3	0.71	48.5	11.7	0.53	0.80[a]

[a] $P < 0.0001$.

higher alpha values than abbreviated scales even if the average item-to-total score correlations are similar.

3.2. Factor structure

In an effort to cross-validate the subscale structure reported by Espie et al. [8], we conducted a factor analysis of the DBAS-10 items using the PC version of the SAS (Statistical Analysis System, Cary, NC) software package. To extract factors or subscales, we used the orthogonal, Varimax rotation method contained in this software. Since the findings of Espie's group were based on data derived solely from insomnia sufferers, we excluded our normal sleeper group from this analysis.

Consistent with Espie et al. [8], our analysis produced three factors which had eigenvalues ≥ 1 and accounted for slightly over half (51.6%) of the total sample variance. Table 2 shows the results of both our factor analysis and the one performed by Espie's group along with the alpha statistics for each respective subscale within each of our three samples. These data show a number of similarities between our factor analysis and the analysis conducted by Espie's group. In both studies, items 10, 12, and 21 loaded highly on Factor 1 and items 5, 8 and 17 loaded highly on Factor 2. However, Espie's group found that items 1 and 2 also loaded most highly on Factor 1 whereas these two items composed our Factor 3. The most notable differences between our results and those of Espie's group were the factor loadings found for items 7 and 22. In the Espie et al. [8] study, these two items clustered together to form Factor 3; we found that item 22 loaded most highly on our second factor whereas item 7 did not load substantially on any of the factors extracted in our sample. Interestingly, our Factors 1 and 2 as well as the first two factors found by Espie's group appear to have acceptable levels of internal consistency as suggested by the alpha values noted in the bottom portion of Table 2. Our third factor appears to have more modest internal consistency whereas the third factor derived by Espie et al. [8] appeared to have, at best, very low internal consistency in our samples.

3.3. Tests of DBAS-10 validity

To test the validity of the DBAS and DBAS-10, we first used a one-way analyses of variance (ANOVAs) to determine if the full DBAS and the DBAS-10 statistically differentiated the insomnia sufferers from the normal sleepers contained in the first research cohort. Our results showed that both of these measures discriminated these two groups of individuals. Our insomnia sufferers (mean DBAS 35.5, SD 10.5) had significantly higher ($F(1, 136) = 28.2$, $P < 0.0001$), or more pathological scores on the full DBAS than did our normal sleepers (mean DBAS 26.4, SD 9.7). Similarly, the DBAS-10 scores of our insomnia sufferers (mean DBAS-10 43.3, SD 15.0) were significantly ($F(1, 136) = 15.9$, $P < 0.0001$) higher than those shown by our normal sleepers (mean DBAS-10 33.5, SD 14.0). Thus, like the standard DBAS, the abbreviated version of this measure appears sensitive to the cognitive differences commonly noted between insomnia sufferers and non-complaining normal sleepers.

We also analyzed data from our second research cohort to determine the sensitivity of the full DBAS and DBAS-10 for detecting decreases in dysfunctional beliefs about sleep resulting from CBT. We

Table 2
Comparison of factor analyses for DBAS-10 in the current and Espie et al. samples

DBAS-10 item number and content	Current study's findings			Espie et al. findings[a]		
	Factor 1	Factor 2	Factor 3	Factor 1	Factor 2	Factor 3
1. I need 8 hours of sleep to feel refreshed and function well during the day	0.202	−0.124	**0.770**	**0.686**	−0.113	0.145
2. When I don't get the proper amount of sleep on a given night, I need to catch up on the next day by napping or on the next night by sleeping longer	−0.015	0.070	**0.796**	**0.669**	−0.077	0.087
5. I am concerned that chronic insomnia may have serious consequences on my physical health	0.326	**0.668**	−0.146	0.163	**0.774**	−0.046
7. When I have trouble getting to sleep, I should stay in bed and try harder	0.087	0.248	0.229	0.103	0.147	**0.873**
8. I am worried that I may lose control over my abilities to sleep	0.237	**0.680**	−0.029	0.125	**0.816**	−0.021
10. After a poor night's sleep, I know that it will interfere with my daily activities on the next day	**0.736**	0.108	0.168	**0.709**	0.316	0.014
12. When I feel irritable, depressed or anxious during the day, it is mostly because I did not sleep well the night before	**0.754**	0.273	−0.018	**0.617**	0.399	−0.101
17. When I sleep poorly on one night, I know it will disturb my sleep schedule for the whole week	0.048	**0.489**	−0.023	−0.056	**0.477**	0.038
21. When I feel tired, have no energy, or just seem not to function well during the day, it is generally because I did not sleep well the night before	**0.790**	−0.015	0.110	**0.707**	0.144	−0.298
22. I get overwhelmed by my thoughts at night and often feel I have no control over this racing mind	−0.194	**0.690**	0.201	0.113	0.404	**−0.454**
Chronbach's alpha values						
Cohort 1: normal sleepers	0.65	0.43	0.35	0.67	0.47	0.00
Cohort 1: insomnia sufferers	0.77	0.55	0.51	0.64	0.58	0.31
Cohort 2: insomnia sufferers	0.60	0.56	0.48	0.53	0.55	0.16

[a] Results of the Espie et al. factor analysis are taken from Ref. [8].

assumed that CBT would produce larger changes/reductions in dysfunctional sleep-related cognitions than would either RT or PC treatments. To test this assumption, we statistically compared the pre-to-post-treatment DBAS and DBAS-10 'change scores' of these three treatment groups using one-way analyses of covariance (ANCOVA) which adjusted for pre-treatment (baseline) differences in DBAS and DBAS-10 scores.

Fig. 1a shows the pre-to-post-treatment DBAS and DBAS-10 changes displayed by our groups of insomnia sufferers treated with either CBT, RT, or the inactive PC. This graph shows that each of the three treatment groups displayed a pre-to-post-treatment decrease in their mean DBAS and DBAS-10 scores. However, results of our ANCOVAs showed that pre-to-post-treatment changes in total DBAS scores were statistically comparable ($F(2, 68) = 2.33,\ P > 0.10$) for all three treatment groups. In contrast, the pre-to-post-treatment DBAS-10 changes did differ statistically across the three treatment groups ($F(2, 68) = 4.69,\ P < 0.025$). Bonferroni-corrected post hoc comparisons, as predicted, showed that the CBT-treated insomnia sufferers showed significantly greater decreases on the DBAS-10 than did those in each of the other two treatment conditions.

We further analyzed our data by testing the pre-to-post treatment changes for each of the subscales derived from both the Espie et al. [8] and our factor analysis. Results for the Espie et al. [8] subscales indicated that significant pre-to-post treatment changes were found for Factors 2 ($F(2, 68) = 3.64$, $P < 0.05$) and Factor 3 ($F(2, 68) = 5.60,\ P < 0.05$) but not for Factor 1. Results for the factors derived

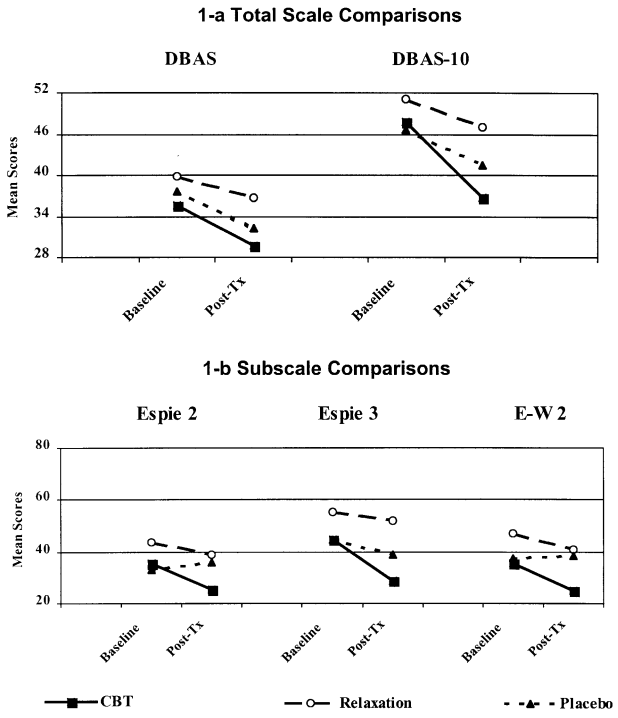

Fig. 1. Pre-to-post-treatment changes on the DBAS, DBAS-10, and DBAS-10 subscales.

from our sample showed that significant changes were found only for Factor 2 ($F(2, 68) = 5.05$, $P < 0.05$). These significant results are displayed in Fig. 1b.

4. Discussion

Despite the increasing interest in and apparent efficacy of cognitive–behavioral insomnia treatments (CBT), there has been relatively little work devoted to the development of brief, reliable, and valid measures of the cognitive changes resulting from such interventions. Given this observation, the current study was conducted to replicate and extend recent promising work by Espie et al. [8] who showed that an abbreviated, 10-item version (DBAS-10) of the Dysfunctional Beliefs and Attitudes About Sleep (DBAS) questionnaire maintains the 'overall thrust' of the full instrument, has reasonable internal consistency, contains factorially pure and conceptually meaningful subscales, and is sensitive to treatment-related cognitive changes manifest by insomnia sufferers receiving CBT. Overall, our findings corresponded well with Espie's group in suggesting this instrument's psychometric integrity and potential utility as a treatment outcome measure.

In reviewing our results, it is noteworthy that our correlational analyses produced results that were very similar to those reported by Espie's group. Specifically, like these previous investigators, we found that the DBAS-10 correlated highly with the full DBAS. This finding supports the impression that the abbreviated DBAS serves as a representative substitute for the original instrument. Also noteworthy is the fact that the DBAS-10 proved to have reasonable internal consistency in our samples. In fact, the alpha coefficients we obtained were similar to the value (i.e. 0.69) reported by Espie's group. As expected, we found that the much longer DBAS had slightly higher alpha values, yet we and Espie's group found the DBAS-10's internal consistency very acceptable for a brief instrument. Also, we found acceptable internal consistency for two of Espie's subscales and two of ours. Espie's Factor 3 and our

Factor 3 were each composed of two items and neither had adequate internal consistency.

In addition to these results, we obtained very promising findings in regard to the DBAS-10's discriminant validity and sensitivity to cognitive changes resulting from CBT. Within our first cohort of well-screened research volunteers, we found that the DBAS-10 discriminated those (i.e. normal sleepers) not expected to have dysfunctional beliefs about sleep from others (i.e. insomnia sufferers) expected to manifest dysfunctional sleep-related cognitions. In fact, in this regard, the DBAS-10 performed about as well as the full DBAS. Furthermore, pre-to-post treatment changes on the DBAS-10 successfully discriminated those who received CBT from those who received other types of behavioral insomnia treatments. Trends toward similar group differences on the full DBAS were noted but these did not reach significance. Admittedly, this finding is not particularly surprising since Espie's group originally selected the DBAS-10 items because they changed significantly from the beginning to the end of CBT treatment. However, our results do confirm the sensitivity of these items to CBT and suggest that the DBAS-10 is a useful metric for the cognitive changes resulting from this intervention.

Although we failed to fully replicate the Espie et al. [8] DBAS-10 factor structure, the two studies' results overlap considerably. Espie's Factor 1 is separated into two factors (Factor 1 and Factor 3) in our analysis. Also, Espie's Factor 2 and our Factor 2 share three items, indicating this subscale is most likely identifying the same underlying construct. Espie's factor analysis generated a third factor which we did not replicate; however, the low internal consistency of this factor calls into question its psychometric adequacy. Furthermore, we found no significant pre-to-post changes among our treatment groups for either Espie's Factor 1 or our Factors 1 or 3, indicating that these factors may not be sensitive to treatment group differences whether they are aggregated into one scale or separated into two. However, we found significant pre-to-post changes for Espie's Factor 2 and our Factor 2, indicating that this factor may be particularly sensitive to changes that occur with cognitive–behavioral insomnia therapy. Finally, Espie's Factor 3 was sensitive to treatment differences, but this scale did not demonstrate internal consistency in our sample.

Interestingly, both studies provide some corroboration for Morin's [6] original proposed subscale structure of the full DBAS. Factors 1 and 2 in both studies appear internally consistent and seem to measure Morin's original themes pertaining to "misattributions about the consequences of insomnia" and "beliefs that sleep is unpredictable and uncontrollable". The differences in items defining these factors in the two studies likely are attributable to marked differences between their participant samples. We enrolled highly screened individuals whereas Espie and colleagues enrolled a more 'real-world' sample of medical clinic patients. Hence, it could be argued that the Espie et al. [8] findings may be more reflective of the DBAS-10 subscale structure for the 'typical' clinical patient. However, the noted differences between the Espie et al. [8] findings and ours suggest that further study of the DBAS-10 subscale structure is warranted.

In considering the current findings, several limitations of this study should be considered. First, it should be noted that only middle-aged and older adults were included in the current study so the results cannot be generalized to younger age groups. In addition, data were gathered from carefully screened research volunteers. Whether the current findings apply to normal sleepers in general or clinical insomnia patients remains untested. It should also be noted that DBAS-10 data were obtained by administering the full DBAS to all of our research volunteers and then extracting DBAS-10 items. Hence, the results reported may not be identical to those obtained by using the abbreviated instrument as a stand-alone measure. Finally, the results presented do not imply that the DBAS-10 measures the only or even necessarily the most important dysfunctional beliefs that contribute to insomnia. Nonetheless, the DBAS-10 is a reasonably brief measure which appears promising for future studies of both the cognitive mechanisms which perpetuate insomnia and the attitudinal changes resulting from CBT treatment.

Acknowledgements

This research is supported by the National Institute of Mental Health Grant MH48187 and Department of Veterans Affairs Merit Review Program.

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